IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant : Neil H. Bander
Serial No. : 09/357,704
Filed : July 20, 1999
Title : TREATMENT AND DIAGNOSIS OF PROSTATE CANCER

Art Unit : 1642
Examiner : Gary Nickol

Commissioner for Patents
Washington, D.C. 20231

DECLARATION UNDER 37 CFR 1.131

I, Neil H. Bander, a citizen of the United States, residing at 2 Hemlock Hills, Chappaqua, NY, 10514, hereby declare as follows:

1. I am the inventor of the subject matter disclosed and claimed in the above-referenced United States Patent Application.

2. I am familiar with the present claims of the application, which are directed to a method of treating, preventing, or delaying development or progression of prostate cancer in a subject.

3. Prior to March 25, 1996, I had conceived my invention as described and claimed in the above-identified application in this country, a NAFTA country or WTO country, and had diligently reduced the invention to practice, as evidenced below.

4. I submit herewith Exhibits A-L, evidence showing conception of the claimed invention prior to the March 25, 1996.

Prior to March 25, 1996, I had conceived of using monoclonal antibodies in the treatment of prostate cancer in humans.

Exhibit A shows an excerpt from a document describing my research on antibodies and their use in cancer that I wrote prior to March 25, 1996. The document dates, dates within the text of the document, and the name of the individual to whom the document is addressed, have been redacted in the excerpt provided in Exhibit A. The document clearly demonstrates that I was actively pursuing monoclonal antibodies for clinical use in prostate cancer.

As indicated in that document, my laboratory had been testing several monoclonal antibodies for their ability to bind to live LNCaP cells, a human prostate cancer model cell line. Several of the antibodies I had already characterized had demonstrated excellent potential, in that they were able to lyse LNCaP cells *in vitro* in the presence of human serum as a source of complement.

I had demonstrated that the antibodies localized to prostate cancer and sites of metastatic disease (in the lymph nodes and liver). More importantly, I had demonstrated that administration of these antibodies to human subjects resulted in treatment and prevention of prostate cancer. For example, I demonstrated that decreased PSA levels were seen upon administration of these antibodies to human subjects, and that there were no signs of relapse seen in these subjects. All of this is shown in Exhibit A. Exhibit A does not discuss the specific antibodies of the invention. In connection with the work described in Exhibit A, I characterized other monoclonal antibodies including the antibodies of the invention. This is shown in Exhibits B-L below.

Exhibit B, is a tag from a mouse cage in my (Dr. Bander's) laboratory indicating that mice immunized with LNCaP cells as part of "Fusion E" experiments were received, immunized, and given a final booster. Dates on the tag have been redacted. The dates show the work was done prior to March 25, 1996.

Exhibits C-L discussed below all show pages from notebooks from my (Dr. Bander's) laboratory. The date on each of the pages is redacted. Each page is dated prior to March 25, 1996.

Exhibit C is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry shows rosette and cytotoxicity studies of the fusion E antibodies including monoclonal antibody E99 (which is an antibody of the invention), and demonstrates that monoclonal antibody E99 binds to LNCaP cells, a human prostate cancer cell line.

Exhibit D is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds renal tubules very weakly, and binds prostate cancer cells strongly.

Exhibit E is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds to benign hyperplastic prostate tissue obtained from various human patients. E99 was detected using a fluorescein label.

Exhibit F is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 is an IgG_3 class antibody.

Exhibit G is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that the E99 antibody is capable of lysing PSMA-expressing LNCaP cells, but not PSMA-negative PC3 and Du145 cells.

Exhibit H is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds strongly to prostate cancer and benign hyperplastic prostate tissues from human patients, binds normal kidney proximal tubules weakly, and does not bind at all to normal liver, lung, pancreas, testis, esophagus, uterus, small bowel, stomach, thyroid, or spleen.

Exhibit I is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99, J415 and J533 (all of which are antibodies of the invention) bind kidney proximal tubules and LNCaP prostate cancer cells, a human prostate cancer cell line, but do not bind normal colon.

Exhibit J is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 and J591 (both of which are antibodies of the invention) bind kidney proximal tubules and LNCaP prostate cancer cells, but do not bind normal colon.

Exhibit K is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that the J415, J533, and E99 antibodies bind weakly to proximal tubule cells of the normal kidney, and bind benign hyperplastic prostate and cancerous prostate tissue from human patients, and that the J415 and J533 antibodies do not bind at all to normal liver, small intestine, or lung.

Exhibit L is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates monoclonal antibodies including E99, J415, J533 and J591 bind to prostate cancer tissue from a number of individuals.

5. Exhibit A demonstrates that I had conceived of using monoclonal antibodies in the treatment and prevention of prostate cancer and Exhibits B-L demonstrate that antibodies which bind PSMA and recognize benign hyperplastic, and cancerous prostate cells from human patients were produced for clinical use in human subjects prior to March 25, 1996. In sum, I submit evidence herewith that shows conception of the claimed invention prior to March 25, 1996.

6. Very shortly after March 25, 1996, namely, May 6, 1996, just a little more than 5 weeks later, the claimed methods were constructively reduced to practice upon filing of provisional application 60/016,976 from which the above-identified application claims priority.

7. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment, under Title 18 § 1001 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

10/7/02
Date

Neil Bander, MD

Exhibit A

[redacted]

Particularly when viewed in the context of the advances in the mAb therapy field in general, the attributes and strengths of mAbs are particularly well-suited to the demands of prostate cancer therapy:

1. mAbs can specifically localize to disseminated tumor sites at levels orders of magnitude higher than normal tissues.

2. Therapeutic efficacy has been proven in tumor types (e.g., colon cancer and NHL) where the clinical setting resembles prostate cancer.

3. mAbs have a number of potential mechanisms of anti-tumor activity including:

   a. the relative radiosensitivity of PCa provides one potential class of cytotoxic agents to specifically deliver to tumor sites by way of mAb.

   b. mAbs can trigger the host's own immune response to tumor.

4. Prostate cancer metastases are small-volume sites (typically measured in microns or mm) ideal for radioisotope or immunotherapy.

5. The availability of established parameters such as PSA and pathological features (e.g., stage, Gleason score, seminal vesicle invasion, positive margins, nodal disease, etc.), provide appropriate indications for adjuvant mAb therapy where such therapy is likely to be most beneficial.

6. Last, but not least, is the fact that mAbs are non-toxic.

[redacted]

We believe that we are well on the way to prove that these advantages are more than just theoretical. We have recently completed our mAb Prost 30 biodistribution study in 15 patients with prostate cancer. Doses ranged from 1.0 to 20.0 mg of mAb. Fourteen of the 15 patients had their prostates *in situ* and were evaluable for localization of Prost 30. In all 14 of these cases, including two with prior radiation therapy, the prostate was successfully imaged. In two cases, patients had known sites of metastatic disease imaged on conventional CT scans: regional lymph nodes (both patients) and liver (1 patient). In these cases, these sites also were successfully imaged with Prost 30. In four cases, after resecting the prostate one week after mAb administration, the prostate specimens were scanned alongside specimens of blood drawn at the time of the resection (see appended representative figure). These studies confirmed specific uptake in the prostate at substantially higher levels than in the blood, and this uptake persists for more than one week. No patient on the trial had any side effects.

[redacted] [redacted]

Having established the ability of the mAb to localize to disseminated sites of disease, more interesting and potentially far more important is the observation that two hormone-refractory patients with progressively rising PSAs prior to entry on the imaging trial responded with substantial (75%) decreases in their PSAs each lasting 10 months after a single 5 mg dose of Prost 30. None of the other patients on the trial are evaluable for response due to receiving other therapy in addition to Prost 30. As the isotope dose used in this biodistribution trial was too low to explain the responses, we believe the responses were due to the mAb triggering an endogenous anti-tumor immune response. Another interesting and provocative observation is that this trial included 6 "high-risk" patients (high PSA ± high Gleason ± high stage) who underwent radical prostatectomy plus Prost 30 treatment. None of these patients have demonstrated signs of relapse (either metastatic disease or (PSA) failure) with a median follow-up of almost 2 years.

[redacted] unconjugated ("naked") Prost 30 in a series of patients evaluable for response. This was to establish the safety of naked antibody-as a prelude to an adjuvant trial similar to that already shown effective in colon cancer -- and to provide a benchmark with which to compare the results of a radiolabeled mAb trial. Doses range from 1.25 mg to 5.0 mg -- the level at which we saw the responses in the earlier trial. Fifteen weeks into this trial we have entered 16 patients. Many of the patients, including some at the lowest dose level, have responded with declines in PSA ranging from 25-55%. It is obviously too early to discuss duration of response. While this data is exceedingly preliminary, it is certainly provocative, particularly as the mAb has no conjugated cytotoxic moiety.

We have also developed in the laboratory a higher affinity Prost 30 mAb which we have designated Prost 130. Prost 130 binds the same antigen as Prost 30, but at a different, and repeated, site. It is possible that these mAbs (Prost 30 and 130) will be additive or synergistic in combination. Two other mAbs we have recently developed, C37 and C219, have demonstrated both prostate specificity (in vitro and in vivo) and the ability to directly lyse LNCaP cells in vitro in the presence of human serum as a source of complement. Furthermore, the cytotoxicity of these mAbs are synergistic when combined in vitro. [redacted] these three mAbs (Prost 130, C37 and C219) have been contracted to an FDA-approved manufacturer for production of clinical grade material for upcoming trials.

[redacted]

Exhibit B

FORMS BY MER LAB & LABEL BOX 147 ALBERTSON N Y 11507
1983

Dr Bowden

[redacted]

Fusion: E

COST CENTER # 6501

CAGE ID # 92080 RM. # [redacted]

INVESTIGATOR [redacted] EXT. 65499

SP/STR. BALB/c SEX ♀

DATE REC'D [redacted] PO # _____

ID # OR #/CAGE _____ VENDOR _____

Dr Lin Peggy

LNCaP cell 6x10⁶/per mouse i.p

date [redacted]

Fusion: E date [redacted] i.p

date [redacted] i.p

ANIMAL MAINTENANCE

CHARGES TO C.C. _____

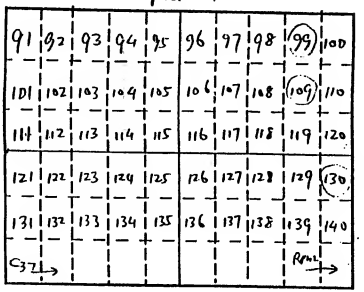
FUND# _____

PROTOCOL# _____

final booster : [redacted]

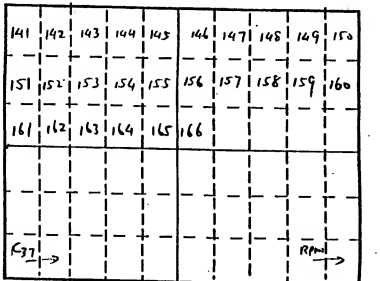
Cytotoxicity for LNCap

plate 1



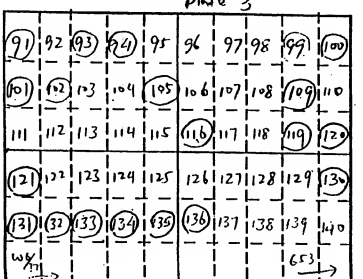
Fusion E

plate 2



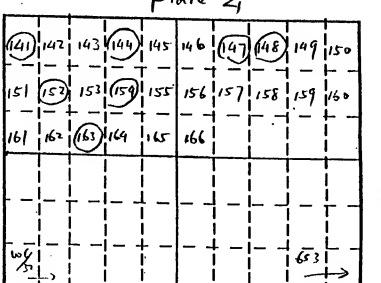
Rosetta test for LNCap

Plate 3



Fusion E

Plate 4



Terasaki Plate

Exhibit C

E. coli D

Slide	Section	MoAb	Result		
1	NK, Neoplasm, Pca	E 91	+	+	+
2	(control)	E 93	+	+	+
3		E 94	+	+	+
✓ 4		E 99	true (+)	−	+
5		E 100	−	−	−
6		E 101	+	+	+
7		E 102	−	−	−
8		E 105	+	+	+
9		E 109	+	+	+
10		E 116	−	+	+
✡ VII		E 119	−	−	+
12		E 120	+	+	+
13		E 121	+	−	+
14		E 130	+	+	+
15		E 131	+	+	+
16		E 132	+	+	+
17		E 133	+	+	+
18		E 134	+	+	+
19		E 135	−	−	−
20		E 136	+	+	+
21		E 141	+	+	+
22		E 144	+	+	+
23		E 147	+	+	+
24		E 148	−	−	−
25		E 152	+	+	+
26		E 154	+	+	+
27		E 163	−	−	−
G 28		Fil 46/2 Pan	+	+	+

Exhibit E

Slide	Section	Mouse		Result	
				epith	stroma
1	BPH (Friedlander)	C37 ascite 1:100	C219 ascite 1:100	—	+
2	"	E27	Eqq		
3	"	653	PA10	—	+
4	"	C37 ascite 1:100	C219 ascite 1:100	—	—
5	"	E27	Eqq	—	—
6	"	653	PA10	—	+
7	BPH (Rodriguez, J)	C37 ascite 1:100	C219 ascite 1:100	epith —	stroma +
8	"	E27	Eqq	+	+
9	"	653	PA10	—	+
10	"	C37 ascite 1:100	C219 ascite 1:100	—	+
11	"	E27	Eqq	+	weak +
12	"	653	PA10	—	+
13	BPH (CHAN, M. Y)	C37 ascite 1:100	C219 ascite 1:100	epith —	stroma +
14	"	E27	Eqq	—	+
15	"	653	PA10	—	+
16	"	C37 ascite 1:100	C219 ascite 1:100	—	—
17	"	E27	Eqq	—	—
18	"	653	PA10	—	+
19	BPH (Deek, F)	C37 asc. 1:100	C219 ascite 1:100	epith —	stroma +
20	"	E27	Eqq	+	+
21	"	653	PA10	—	+
22	"	C37 ascite 1:100	C219 ascite 1:100	—	+
23	"	E27	Eqq	+	+
24	"	653	PA10	—	+

FITC-RAMS (slides 4–6)

FITC-RAMS (slides 10–11)

FITC-RAMS (slides 16–18)

FITC-RAMS (slides 22–23)

EXHIBIT F

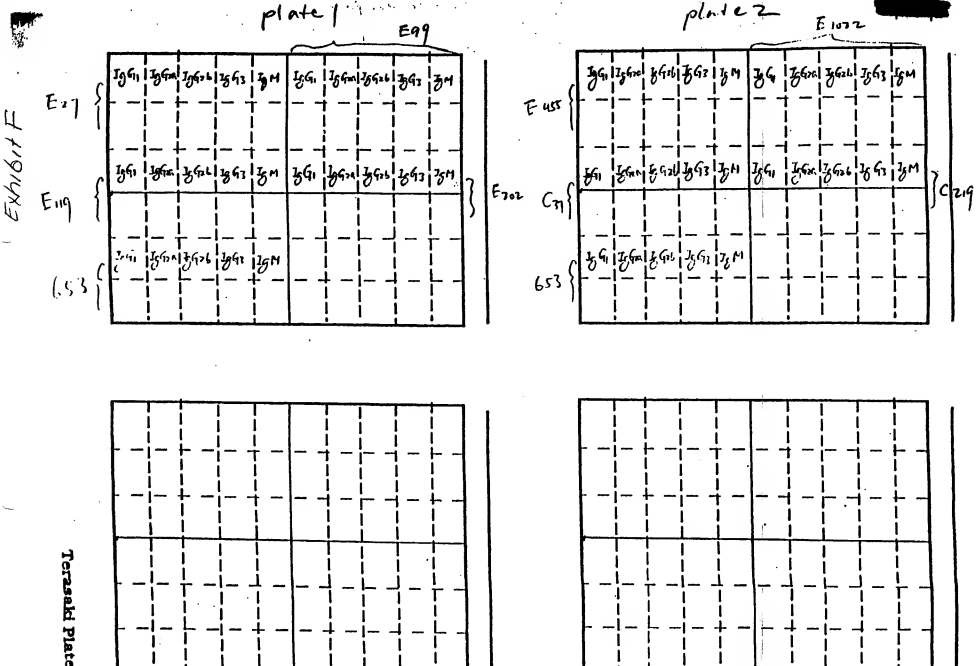


plate 1
E99
E27
E119
653
E702
plate 2
E 1072
E 455
C 71
653
C219
Terzaki Plate

Ex. 16 of F (cont'd)

[redacted]

plate 1 — 15 min

		1 $\frac{1}{2}G_1$	2 $\frac{1}{2}G_{12a}$	3 $\frac{1}{2}G_{12b}$	4 $\frac{1}{2}G_3$	5 $\frac{1}{2}M$	6 $\frac{1}{2}G_1$	7 $\frac{1}{2}G_{12a}$	8 $\frac{1}{2}G_{12b}$	9 $\frac{1}{2}G_3$	10 $\frac{1}{2}M$	15min plate 1	
E27	F	+0.22	+0.21	+0.18	+0.34	+1.41	+0.23	+0.21	+0.17	+0.92	+0.58	E99	F
	E	+0.23	+0.22	+0.17	+0.30	+1.37	+0.26	+0.22	+0.18	+1.11	+0.51		E
E119	D	+0.94	+0.19	+0.19	+0.35	+1.39	+1.00	+0.71	+0.31	+0.31	+1.41	E202	D
	C	+0.85	+0.21	+0.20	+0.32	+1.41	+0.95	+0.23	+0.30	+0.40	+1.47		C
653	B	+0.21	+0.17	+0.18	+0.23	+0.26	+0.09	+0.10	+0.10	+0.15	+0.11		B
	A	+0.18	+0.17	+0.13	+0.26	+0.30	+0.09	+0.10	+0.10	+0.13	+0.10		A

plate 2 — 15 min

		1 $\frac{1}{2}G_1$	2 $\frac{1}{2}G_{12a}$	3 $\frac{1}{2}G_{12b}$	4 $\frac{1}{2}G_3$	5 $\frac{1}{2}M$	6 $\frac{1}{2}G_1$	7 $\frac{1}{2}G_{12a}$	8 $\frac{1}{2}G_{12b}$	9 $\frac{1}{2}G_3$	10 $\frac{1}{2}M$	15min plate 2	
E455	F	+0.30	+0.26	+0.22	+0.36	+1.43	+0.38	+0.26	+0.22	+0.39	+1.41	E202	F
	E	+0.24	+0.23	+0.21	+0.48	+1.43	+0.43	+0.25	+0.17	+0.35	+1.40		E
C37	D	+0.29	+0.27	+0.20	+0.34	+1.43	+0.30	+0.28	+0.20	+0.32	+1.42	C219	D
	C	+0.28	+0.25	+0.19	+0.34	+1.43	+0.25	+0.23	+0.20	+0.32	+1.40		C
653	B	+0.26	+0.19	+0.14	+0.26	+0.32	+0.10	+0.11	+0.09	+0.12	+0.13		B
	A	+0.24	+0.21	+0.13	+0.27	+0.31	+0.09	+0.12	+0.09	+0.29	+0.09		A

plate 1 — 60 min

		1 $\frac{1}{2}G_1$	2 $\frac{1}{2}G_{12a}$	3 $\frac{1}{2}G_{12b}$	4 $\frac{1}{2}G_3$	5 $\frac{1}{2}M$	6 $\frac{1}{2}G_1$	7 $\frac{1}{2}G_{12a}$	8 $\frac{1}{2}G_{12b}$	9 $\frac{1}{2}G_3$	10 $\frac{1}{2}M$	60min plate 1	
E27	F	+0.48	+0.46	+0.36	+0.81	+1.41	+0.50	+0.46	+0.28	+1.38	+1.19	E99	F
	E	+0.52	+0.46	+0.36	+0.72	+1.43	+0.60	+0.47	+0.32	+1.41	+1.16		E
E119	D	+1.39	+0.41	+0.41	+0.80	+1.43	+1.39	+0.67	+0.52	+0.77	+1.41	E202	D
	C	+1.42	+0.47	+0.42	+0.72	+1.44	+1.35	+0.55	+0.41	+0.75	+1.43		C
653	B	+0.45	+0.33	+0.27	+0.53	+0.61	+0.13	+0.14	+0.14	+0.13	+0.15		B
	A	+0.38	+0.35	+0.24	+0.58	+0.71	+0.12	+0.14	+0.13	+0.20	+0.13		A

plate 2 — 60 min

		1 $\frac{1}{2}G_1$	2 $\frac{1}{2}G_{12a}$	3 $\frac{1}{2}G_{12b}$	4 $\frac{1}{2}G_3$	5 $\frac{1}{2}M$	6 $\frac{1}{2}G_1$	7 $\frac{1}{2}G_{12a}$	8 $\frac{1}{2}G_{12b}$	9 $\frac{1}{2}G_3$	10 $\frac{1}{2}M$	60min	
E455	F	+0.66	+0.56	+0.48	+0.85	+1.41	+0.88	+0.60	+0.44	+0.92	+1.47	E202	F
	E	+0.56	+0.49	+0.43	+1.04	+1.41	+0.94	+0.54	+0.34	+0.80	+1.45		E
C37	D	+0.66	+0.64	+0.41	+0.79	+1.41	+0.68	+0.63	+0.41	+0.73	+1.43	C219	D
	C	+0.61	+0.54	+0.37	+0.77	+1.44	+0.58	+0.53	+0.42	+0.79	+1.44		C
653	B	+0.55	+0.38	+0.25	+0.55	+0.72	+0.12	+0.15	+0.12	+0.18	+0.20		B
	A	+0.51	+0.42	+0.24	+0.59	+0.69	+0.11	+0.17	+0.10	+0.33	+0.10		A

Exhibit G

Cytotoxicity Test

E27 E99 E109 E202 E255 E1022 C37 C219 653

Pc3

Du145

LNCap

Cytotoxicity to LNCap [redacted]

	Guinea Pig serum								
C37	+ 1:3	+ 1:3	+ 1:4	+ 1:4	+ 1:5	+ 1:5	+ 1:6	+ 1:6	653 1:3	653 1:5
C219	Guinea Pig serum									
	+ 1:3	+ 1:3	+ 1:4	+ 1:4	+ 1:5	+ 1:5	+ 1:6	+ 1:6	653 1:3	653 1:5
C37	Human Serum									
	+ 1:3	+ 1:3	+ 1:4	+ 1:4	+ 1:5	+ 1:5	+ 1:6	+ 1:6	653 1:3	653 1:5
C219	Human Serum									
	+ 1:3	+ 1:3	+ 1:4	+ 1:4	+ 1:5	+ 1:5	+ 1:6	+ 1:6	653 1:3	653 1:5
New C37	+ 1:1	+ 1:2	+ 1:4	+ 1:8	60% 1:16	1:32	1:64	1:128	653	653
New C219	+ 1:2	80% 1:2	40% 1:4	1:8	1:16	1:32	1:64	1:128	653	653

Terasaki Plate

EXHIBIT H

[redacted]

Slide	Section	MoAb		Result	
1	NK (unsoeld, W),	E27	E99	—	PT weak +
2		E202	E455	—	—
3		653	F31	—	+
4	Nor liver	E27	E99	—	—
5	(31-53291)	E202	E455	—	—
6		653	WK/32	—	+++
7	Nor lung (208)	E27	E99	—	—
8		E202	E455	Alveola (-)	Bronchiole Basal (+)
9		653	WK/2	—	+++
10	Nor pancreas (271)	E27	E99	+++	—
11		E202	E455	—	—
12		653	WK/32	—	+++
13	Nor Testis (259)	E27	E99	—	—
14		E202	E455	—	—
15		653	WK/32	—	connect w/ tissue ++ —
16	Pca (WHEELER, A)	E27	E99	hetrogen +++	weak +
17		E202	E455	basal + — ++	basal +++
18		653	P410	—	weak +
19	Pca (...)	E27	E99	hetrogen + — ++	+ — +
20	Culver	E202	E455	hetrogen basal +	hetrogen basal + —
21		653	P410	—	+
22	BPH (...)	E27	E99	—	+
23	Chanlwonsam	E202	E455	hetrogen +	hetrogen ++
24		653	P410	—	++

216
35
41
39
34
52
51

(Exhibit H (cont'd)

[redacted]

	Slide	Section	MoAb			
	1	Esophagus (31-57925)	E27	E99	—	—
	2		E202	E455	basal +	basal +
6	3		653	WG/32	—	+
	4	Uterus (31-57931)	E27	E99	—	—
6	5		E202	E455	—	—
	6		653	WG/32	—	+
	7	small bowel (31-51650)	E27	E99	+/-	—
	8		E202	E455	—	—
6	9		653	WG/32	—	+
	10	Stomach (31-53027)	E27	E99	—	—
8	11		E202	E455	section not good	
	12		653	WG/32	+	+
	13	Thyroid (P91-422)	E27	E99	—	—
	14		E202	E455	—	—
11	15		653	WG/32	—	+
	16	spleen (P91-39)	E27	E99	+?	—
	17		E202	E455	—	—
11	18		653	WG/32	—	+
	19	PCa (Mahoney, W)	E27	E99	heterogen	+
	20		E202	E455	+	+/-
5-9	21		653	P910	—	heterogen +/-
	22	Pca verdugo, J	E27	E99	—	+-+
6,7	23		E202	E455	heterogen weak +	heterogen +
	24		653	P910	—	+-+

Exhibit I

Slide	Section	MoAb	Result		
✱ 1	NK, N.C., LNCap tumor	J-267	+	−	+
2		J-271	+	+	+
3		J-284	+	+	+
4		J-297	+	−	−
5		J-331	+	+	+
6		J-377	+	+	+
7		J-380	+	−	−
8		J-382	+	+	+
9		J-385	+	+	+
10		J-386	−	+	−
11		J-407	+	+	+
✱ 12		J-415	PT+	−	+
13		J-421	+	+	+
14		J-437	+	+	+
15		J-445	+	+	−
16		J-457	+	+	+
17		J-472	−	−	−
18		J-478	−	−	−
19		J-518	−	−	−
20		J-515	−	−	−
21		J-527	+	−	+
✱ 22		J-533	PT+	−	+
23		653	−	−	−
24		Eag	OT+	−	+

Exhibit J

Strip	Section	MDAU				
1	NK (Rico, Russell)	J-4	J-5	+		+
2	"	J-14	J-96	+		+
12 3	"	J-110	J-128	+		+
4	"	J-254	J-262	—		—
5	"	653	E31	—		+
6	NK, River (731-53291)	J-4	J-5	+		+
7		J-14	J-96	+		+
3 8		J-110	J-128	+		+
9		J-254	J-262	—		—
10		653	E92	—		+
11	PCA (Lee, Jim)	J-4	J-5	+		+
12		J-14	J-96	+		+
13		J-110	J-128	+		+
3 14		J-254	J-262	—		—
15	"	653	Egg	—		+
V 16	NK, N.C. [illegible]	J-4	J-5	+	—	+
17	"	J-662		+	—	+
18	"	J-722		—	—	—
19	"	J-794		+	+	+
20	"	J-797		—	+	+
21	"	J-827		—	—	—
22	"	J-832		+	—	—
23	"	J-816		+	+	+
24	"	J-823		+	+	+
25	"	653		—	—	—
26	"	Egg		P+	—	+

Exhibit K

	slide	Section	MoAb		Result	
	1	NK (RITA Ronald)	J-415	J-533	PT+	PT+
12	2	"	663	Egg	—	PT+
35	3	Nor River (31-53291)	J-415	J-533	—	—
	4	Nor Sm Intestine	J-415	J-533	—	—
44	5	(LAERANCE E)	653	W6/32	—	+
	6	Nor Lung (2UE)	J-415	J-533	—	—
41	7	"	653	W6/32	—	+
	8	BpH (Krammer A)	J-415	J-533	1-3+	1-2+
39	9	"	653	Egg	—	1-2+
59	10	BpH (Jseng David	J-415	J-533	1-3+	1-2+
	11	"	653	Egg	—	1-3+
	12	Pca (Walsh J	J-415	J-533	2-4+	2-4+
65	13	"	653	Egg	—	2-4+
	14	Pca (Linnan E)	J-415	J-533	1-3+	1-3+
63	15	"	653	Egg	—	1-3+

Exhibit L

Slide	Section	MoAb		Result	
1	PCa	E455	E99	0-2+	0-2+
2	(CANNON, THOMAS)	J-415	J-533	0-3+	0-2+
3		J-591	653	0-3+	—
4		H125	H647	—	0-3+
5		H648	H108-3	0-3+	0-1+
6		P410	P30	1+	3+
7	PCa	E455	E99	—	3+
8	(CHIEN, VEN)	J-415	J-533	3+	3+
9		J-591	653	3+	—
10		H125	H647	—	1-3+
11		H648	H108-3	1-3+	2+
12		P410	P30	2+	3+
13	PCa	E455	E99	3+	2-3+
14	(HOPE, ABORTUNADO)	J-415	J-533	1-2+	1-2+
15		J-591	653	1-2+	—
16		H125	H647	0-3+	0-2+
17		H648	H108-3	0-2+	0-1+
18		P410	P30	1-2+	3+
19	PCa	E455	E99	0-1+	1-2+
20	(CHAN YU CHING)	J-415	J-533	1-2+	1-2+
21		J-591	653	1-2+	—
22		H125	H647	occasional 1+	3+
23		H648	H108-3	3+	1+
24		P410	P30	1-2+	3+
25	PCa	E455	E99	4+	0-2+
26	(O'BRIEN, WILLIAM)	J-415	J-533	0-3+	0-3+
27		J-591	653	0-2+	—
28		H125	H647	0-3+	0-3+